We claim:

1. A method for preventing and/or treating an estrogen-dependent disorder selected from endometriosis, uterine fibroids, dysfunctional uterine bleeding, endometrial hyperplasia, polycystic ovarian syndrome, fibrocystic breast disease and fibrocystic mastopathy in a female mammal in need of the prevention, comprising administering an effective amount of exemestane to the mammal.

2. A method according to claim 1, further comprising administering at least one additional therapeutic agent to the mammal.

3. A method according to claim 2, wherein the at least one additional therapeutic agent is selected from the group consisting of danazol, a COX-2 inhibitor, a non-steroidal anti-inflammatory compound (NSAID), a retinoid compound, a matrix metallo-protease inhibitor, an anti-estrogen, GnRH agonist or antagonist, a selective progestin receptor modulator (SPRM) and an angiogenesis inhibitor, or a mixture thereof.

4. A method according to claim 2, wherein the at least one additional therapeutic agent comprises from 2 to 4 subtances selected from the group consisting of danazol, a COX-2 inhibitor, a non-steroidal anti-inflammatory compound (NSAID), a retinoid compound, a matrix metallo-protease inhibitor, an anti-estrogen, GnRH agonist or antagonist, a selective progestin receptor modulator (SPRM) and an angiogenesis inhibitor.

5. A method according to claim 3, wherein the at least one additional therapeutic agent is anti-estrogen, which is a SERM devoid of uterotrophic activity.

6. A method according to claim 5, wherein the SERM is selected from the group consisting of tamoxifen, toremifene, arzoxifene, idoxifene, EM 800, fulvestrant and droloxifene.

7. A method according to claim 3, wherein the at least one additional therapeutic agent is a GnRH agonist selected from the group consisting of leuprorelin, dislorelin, triptorelin, buserelin, nafarelin, goserelin, avorelin, histerelin, compound PTL 03301, compound AN 207, compound TX 397, compound AN 201 and compound SPD 424, or a pharmaceutically acceptable salt thereof.

8. A method according to claim 7, wherein the GnRH agonist is selected from the group consisting of triptorelin, goserelin and leuprorelin or a pharmaceutically acceptable salt thereof.

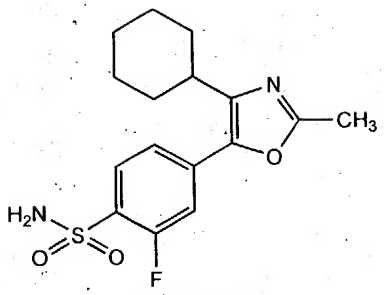
9. A method according to claim 8, wherein the GnRH agonist is triptorelin pamoate.

10. A method according to claim 3, wherein the GnRH antagonist is selected from the group consisting of cetrorelix, abarelix, ramorelix, teverelix, ganirelix, compounds A 75998 and A 84861, compound PM-OV-92, GnRH immunogen, compound D 26344, compound T 98475 and compound MI 1544, or a pharmaceutically acceptable salt thereof.

11. A method according to claim 10, wherein the GnRH antagonist is abarelix or a pharmaceutically acceptable salt thereof.

12. A method according to claim 3, wherein the at least one additional therapeutic agent is a SPRM, which is dienogest or a pharmaceutically acceptable salt thereof.

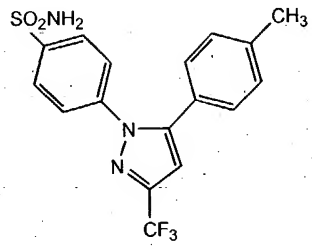
13. A method according to claim 3, wherein the at least one additional therapeutic agent is a COX-2 inhibitor selected from the group consisting of:



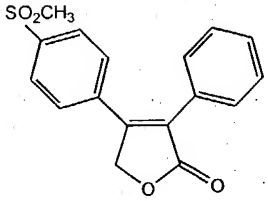
compound A;

5-chloro-3-(4-(methylsulfonyl)phenyl)-2-(methyl-5-pyridinyl) pyridine;

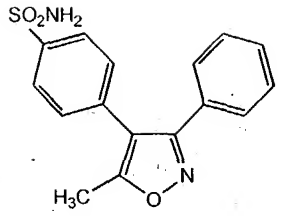
2-(3, 5-difluorophenyl)-3-4(methylsulfonyl)phenyl)-2-cyclopenten-1-one;



compound B;

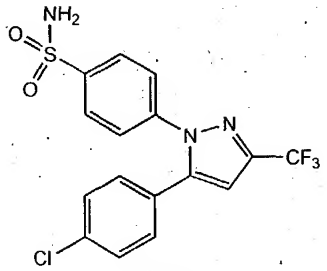


compound C;

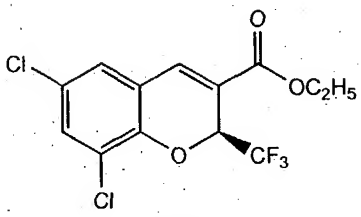


compound D;

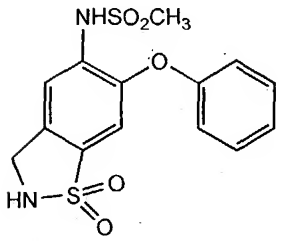
N-[[4-(5-methyl-3-phenylisoxazol-4yl] phenyl]sulfonyl] propanamide;



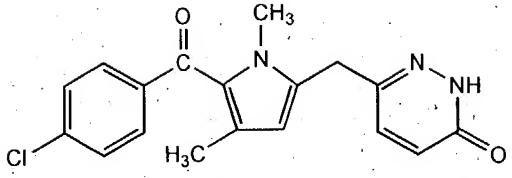
compound E;



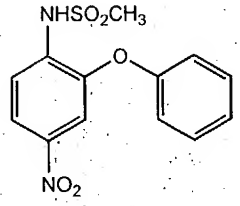
compound F;



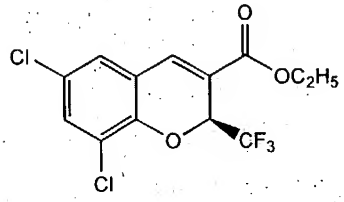
compound G;



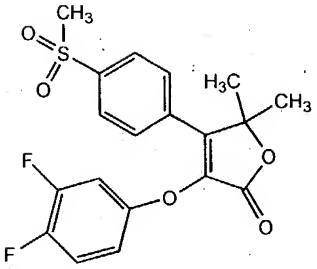
compound H;



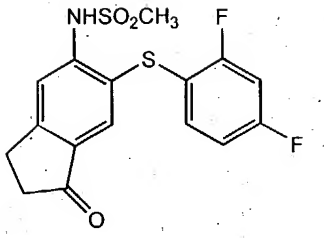
compound I;



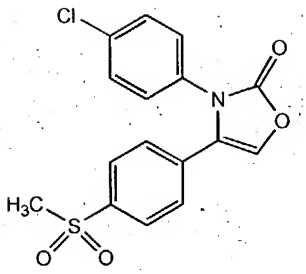
compound J;



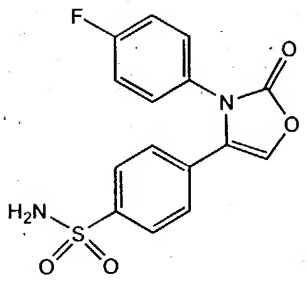
compound K;



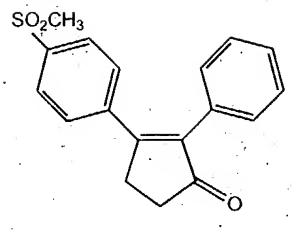
compound L;



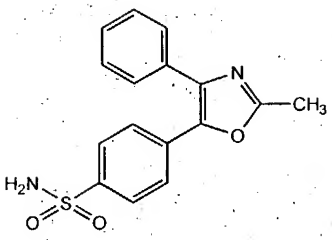
compound M;



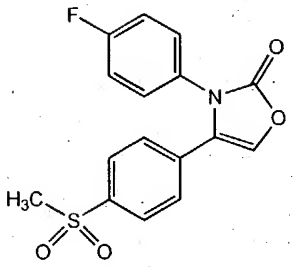
compound N;



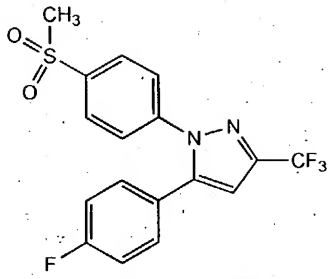
compound O;



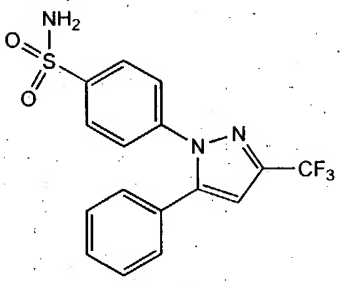
compound P;



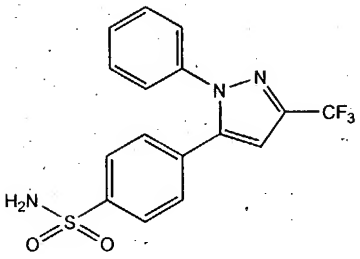
compound Q;



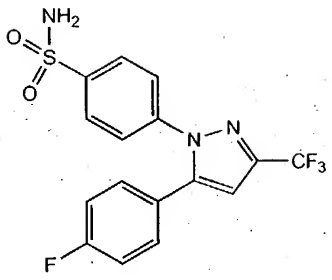
compound R;



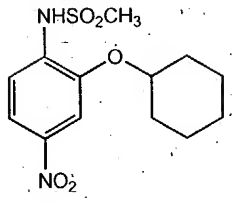
compound S;



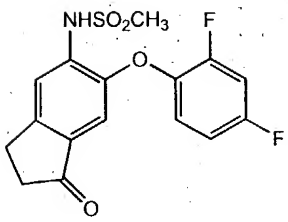
compound T;



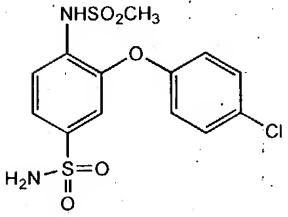
compound U;



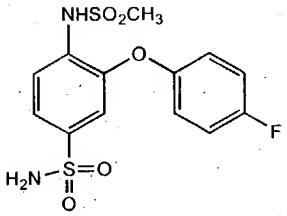
compound V;



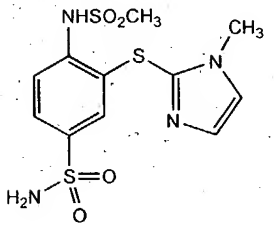
compound W;



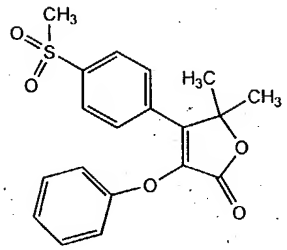
compound X;



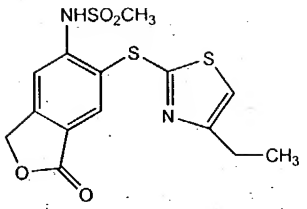
compound Y;



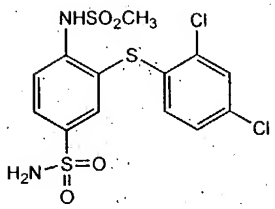
compound Z;



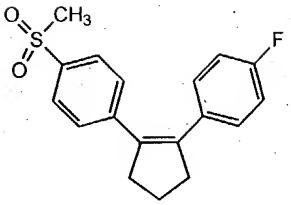
compound AB;



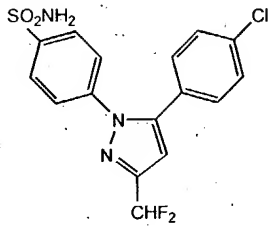
compound AC;



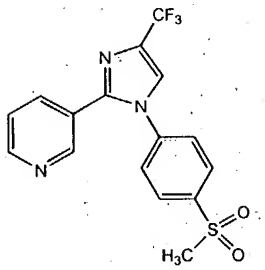
compound AD;



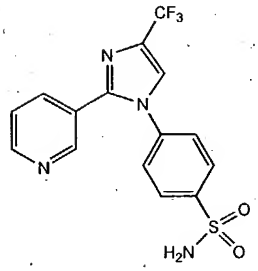
compound AE;



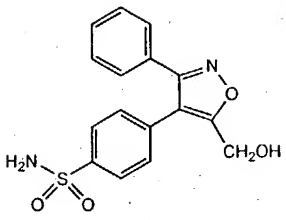
compound AF;



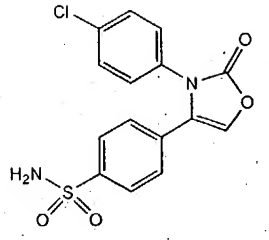
compound AG;



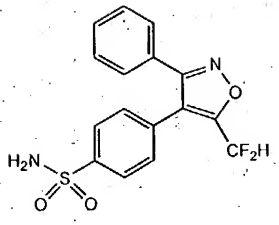
compound AH;



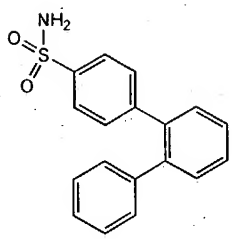
compound AI;



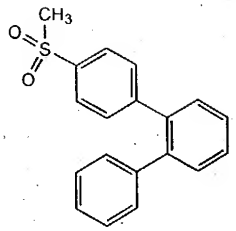
compound AJ;



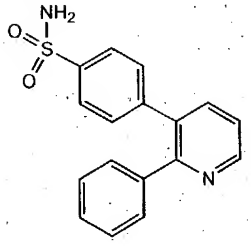
compound AK;



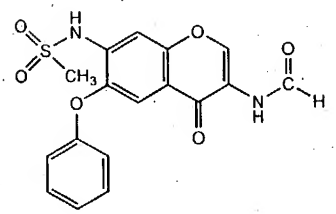
compound AL;



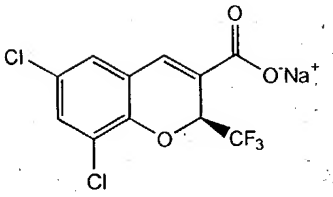
compound AM;



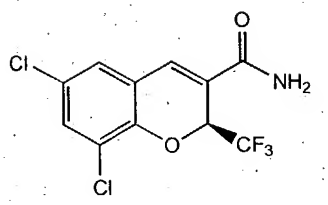
compound AN;



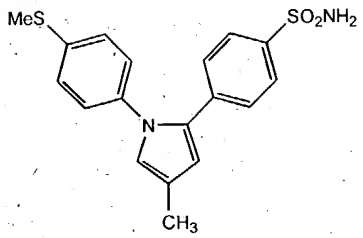
compound AO;



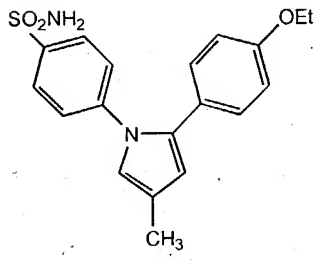
compound AP;



compound AQ;



compound AR;



compound AS;

compound T 614; darbufelone; compound L745337; celecoxib; compound CT3; rofecoxib; compound L783003; compound JT3 522; compound 754; parecoxib; compound S2474; compound LAS 33815; valdecoxib; and compound MK 663.

14. A method according to claim 3, wherein the at least one additional therapeutic agent is a COX-2 inhibitor selected from celecoxib, rofecoxib, parecoxib and valdecoxib.

15. A method according to claim 14, wherein the COX-2 inhibitor is celecoxib.

16. A method according to claim 3, wherein the at least one additional therapeutic agent is a NSAID selected from the group consisting of acetyl salicylic acid, indometacin, sulindac, phenylbutazone, diclofenac, fentiazac, ketorolac, piroxicam, tenoxicam, mecoxicam, meloxicam, cinnoxicam, ibufenac, ibuprofen, naproxen, ketoprofen, nabumetone, niflumic acid and nimesulide, or a pharmaceutically acceptable salt thereof.

17. A method according to claim 16, wherein the NSAID is selected from the group consisting of diclofenac, piroxicam, tenoxicam, mecoxicam, meloxicam, ibufenac, ibuprofen, naproxen and ketoprofen, or a pharmaceutically acceptable salt thereof.

18. A method according to claim 3, wherein the at least one additional therapeutic agent is a retinoid compound selected from the group consisting of Accutane; Adapalene; AGN-193174; AGN-193676; AGN-193836; AGN-193109; AR-623; BMS-181162; CD-437; ER-34617; Etrinate; Fenretinide; Ligand LGD-1550; lexacalcitol; MX-781; mofarotene; MDI-101; MDI-301; MDI-403; Motretinide; 4-(2-[5-(4-methyl-7-ethylbenzofuran-2-yl)pyrrolyl])benzoic acid; N-[4-[2-thyl-1-(1H-imidazol-1-yl)butyl]phenyl]-2-benzothiazolamine; Soriatane; SR-11262; Tocoretinate; Advanced Polymer Systems trans-retinoic acid; UAB-8; Tazorac; TopiCare; TAC-101; and Vesanoid.

19. A method according to claim 3, wherein the at least one additional therapeutic agent is a metallo-protease inhibitor selected from the group consisting of:

1-cyclopropyl-N-hydroxy-4-[[4-[4-(trifluoromethoxy)phenoxy]phenyl]sulfonyl]-4-piperidinecarboxamide monohydrochloride;

N-hydroxy-1-(phenylmethyl)-4-[[4-[4-(trifluoromethoxy)phenoxy]-1-piperidinyl]sulfonyl]-4-piperidinecarboxamide monohydrochloride;

N-hydroxy-1-(pyridinylmethyl)-4-[[4-[4-(trifluoromethyl)phenoxy]phenyl]sulfonyl]-4-piperidinecarboxamide dihydrochloride;

N-hydroxy-2,3-dimethoxy-6-[[4-[4-(trifluoromethyl)phenoxy]-1-piperidinyl]sulfonyl]-benzamide;

N-hydroxy-1-(4-pyridinylmethyl)-4-[[4-[4-(trifluoromethyl)phenoxy]phenyl]sulfonyl]-4-piperidinecarboxamide dihydrochloride;

N-hydroxy-1-(3-pyridinylmethyl)-4-[[4-[4-(trifluoromethyl)phenoxy]phenyl]sulfonyl]-4-piperidinecarboxamide dihydrochloride;

N-hydroxy-1-(2-pyridinylmethyl)-4-[[4-[4-(trifluoromethyl)phenoxy]phenyl]sulfonyl]-4-piperidinecarboxamide monohydrochloride;

BB-2516 (marimastat), N4-[2,2-dimethyl-1-[(methylamino)carbonyl]- propyl]-N1,2-dihydroxy-3-(2-methylpropyl)-, [2S-[N4(R*), 2R*, 3S*]]-);

BMS 275291;

Bay-12-9566 (tanomastat), 4-[(4'-chloro[1,1-diphenyl]-4-yl)oxy]-2-[(phenylthio) methyl] butanoic acid;

AG-3340, N-hydroxy-2,2'-dimethyl-4-[[4-(4-pyridinyloxy)phenyl]sulfonyl]-3-thiomorpholine-carboxamide;

CMT-3 (metastat), 6-demethyl-6-deoxy-4-dedimethylaminotetracycline, batimastat (BB-94); and

D-2163,2-[1S-([(2R,S)-acetylmercapto-5-phthalimido]pentanoyl-L-leucyl)amino-3-methylbutyl]imidazole.

20. A method according to claim 3, wherein the at least one additional therapeutic agent is an angiogenesis inhibitor selected from the group consisting of an $\alpha v\beta 3$ integrin inhibitor, a

protein kinase inhibitor, angiostatin, platelet factor 4 (endostatin), a VEGF inhibitor and thalidomide.

21. A method according to claim 20, wherein the angiogenesis inhibitor is thalidomide.

22. A method according to claim 20, wherein the αvβ3 integrin inhibitor is selected from the group consisting of:

Vitaxin antibody (Ixsys); Merck KgaA EMD-121974, cyclo[RGDF-N(Me)V-];

(10S)-10,11-dihydro-3-[3-(2-pyridinylamino)propoxy]-5H-dibenzo[a,d]cycloheptene-10-acetic acid;

(2S)-7-[[(1H-benzimidazol-2-ylmethyl)methylamino]carbonyl]-2;3,4,5-tetrahydro-4-methyl-3-oxo-1H-1,4-benzodiazepine-2-acetic acid;

(2S)-2,3,4,5-tetrahydro-4-methyl-7-[[[(5-methyl-1H-imidazo[4,5-b]pyridin-2yl]methyl]amino] carbonyl]-3-oxo-1H-1,4-benzodiazepine-2-acetic acid;

(bR)-b-[[[(3R)-2-oxo-3-[2-(5,6,7,8-tetrahydro-[1,8]-naphthyridin-2-yl)ethyl]1-1-pyrrolidinyl] acetyl]amino]-d-(1H-indol-3-yl)pentanoic acid; and

(3R)-N-[3-hydroxy-5-[(1,4,5,6-tetrahydro-5-hydroxy-2-pyrimidinyl)amino]benzoyl]-glycyl-3-(3-bromo-5-chloro-2-hydroxyphenyl)-b-alanine (compound SD 7784).

23. A method according to claim 20, wherein the protein kinase inhibitor is selected from compound SU6668 (3-[4-(2-carboxyethyl-3,5-dimethylpyrrol-2-yl)methylidenyl]-2-indolinone), and compound SU5416 (3-[(2,4-dimethylpyrrol-5-yl)methylidenyl]-2-indolinone).

24. A method according to claim 20, wherein the VEGF inhibitor is selected from the group consisting of compound SU 6668, compound SU 5416, rhuMAbVEGF and compound DC 101.

25. A method according to claim 1, wherein the mammal is a human.

26. A pharmaceutical composition for preventing and/or treating an estrogen-dependent disorder, said preparation comprising exemestane and at least one additional therapeutic agent.

27. The composition according to claim 26, wherein the at least one additional therapeutic agent is selected from the group consisting of danazol, a COX-2 inhibitor, a non-steroidal anti-inflammatory compound (NSAID), a retinoid compound, a matrix metallo-protease inhibitor, an anti-estrogen, GnRH agonist or antagonist, a selective progestin receptor modulator (SPRM) and an angiogenesis inhibitor, or a mixture thereof.

28. A method for treating infertility in a female mammal in need of the treatment, comprising administering an infertility treating effective amount of exemestane to the mammal.

29. A method according to claim 28, wherein the infertility is an estrogen-dependent disorder.

30. A method according to claim 28, wherein the infertility is caused by or associated with endometriosis.

31. A method according to claim 28, wherein the infertility is caused by or associated with polycystic ovarian syndrome.

32. A method according to claim 28, wherein the infertility is anovulatory infertility.

33. A method according to claim 28, wherein the mammal has hypogonadotropic hypogonadism.

34. A method according to claim 28, wherein the mammal has a menstrual cycle disorder.

35. A method according to claim 28, wherein the mammal is a human.

36. A method according to claim 28, wherein exemestane is administered in day 5 to day 7 of a menstrual cycle of the mammal and then stopped.

37. A method according to claim 28, wherein exemestane is administered throughout a menstrual cycle of the mammal and then stopped.

38. A method according to claim 28, wherein the effective amount is a therapeutically effective follicular stimulating amount.

39. A method according to claim 28, wherein the mammal is a candidate for an assisted reproduction technique.

40. A method for inducing ovarian follicular stimulation in a female mammal in need of the induction, comprising administering a therapeutically effective follicular stimulating and/or inhibitory amount of exemestane to the mammal, followed by a stoppage of the exemestane administration, wherein the stoppage provides rebound hyperstimulation of the ovaries.

41. A method according to claim 40, wherein the mammal is a human.